801302

The Evoked Potential: An Experimental Method for Biomechanical Analysis of Brain and Spinal Injury

A. Sances, Jr., R. Weber, J. Myklebust, J. Cusick, S. Larson, P. Walsh, T. Christoffel and C. Houterman
Department of Neurosurgery
The Medical College of Wisconsin and
VA Medical Center

C. Ewing and D. Thomas
Naval Biodynamics Laboratory
New Orleans, Louisiana

B. Saltzberg
Texas Research Institute of Mental Sciences
Houston, Texas

Twenty-Fourth Stapp Car Crash Conference

801302

The Evoked Potential: An Experimental Method for Biomechanical Analysis of Brain and Spinal Injury

A. Sances, Jr., R. Weber, J. Myklebust,
J. Cusick, S. Larson, P. Walsh,
T. Christoffel and C. Houterman
Department of Neurosurgery
The Medical College of Wisconsin and
VA Medical Center

C. Ewing and D. Thomas
Naval Biodynamics Laboratory
New Orleans, Louisiana

B. Saltzberg
Texas Research Institute of Mental Sciences
Houston, Texas

Abstract

Axial forces were applied between the shoulders and the skull of eight male Macaca mulatta monkeys. Forces from 556 to 1444 Newtons produced marked changes in blood pressure, heart rate and distraction of the cervical spinal column with minimal ligamentous disruption. Somatosensory evoked potentials recorded at the cortical and thalamic levels following dorsal column or peripheral nerve stimulation were altered prior to or during changes in heart rate or blood pressure. Similar findings were observed in the efferent responses recorded from electrodes placed on the thoracic spinal cord following stimulation of sensorimotor cortex. Studies in four monkey cadaveric isolated cervical column preparations indicate that disruption occurs with axial loads which are approximately one-third of the maximum used in the in vivo studies.

0585-086X/80/1015-1302/$02.50

THE EVOKED POTENTIAL IS A NEUROELECTRIC RESPONSE to a purposeful physical stimulus. The evoked response has long been a tool of physiology. The first evoked potential was recorded by Caton in 1875, when the Thompson galvanometer detected a current on the surface of the rabbit brain (1).* The somatosensory evoked potential is generated by a current stimulus applied to a peripheral nerve or the central nervous system. Recording is done at a connecting region to determine the integrity of the nervous structure.

The object of this study is to apply the evoked potential to the experimental evaluation of cerebral and spinal cord injury. The evoked potential has proven useful in the evaluation of patients with head trauma (2-5). It has been advanced that the early component of the somatosensory evoked potentials is a measure of the integrity of the brainstem (6,7). In patients with head trauma and evidence of thalamocortical dysfunction, the early somatosensory evoked potential, recorded from the parietal scalp to the ear, was the only component remaining (3,4).

Recent studies have demonstrated that the evoked potential recorded from cerebral cortex or rostral spinal cord secondary to peripheral stimulation in the monkey was immediately reduced in amplitude both by pathologic distraction and pathologic flexion of the thoracic vertebral column (8). Cerebral responses were lost within two minutes after complete occlusion of the ascending aorta, while the responses recorded from the spinal cord persists for approximately ten minutes and then gradually disappears. However, the immediate flexion and distraction responses were not altered. These findings suggest that mechanical trauma alters the spinal cord evoked potentials and that it should be possible to differentiate a mechanical from a vascular insult (8). Clinical studies have demonstrated that the evoked potential is altered in patients with distorted cord and roots displaced by bone and disc. Reconstruction of the spinal canal was followed by a return of the evoked potential with improved function (9). Experimental cervical cord injury in the monkey alters the evoked potential (10). While some reports suggest that the somatosensory evoked potentials travel over several spinal afferent pathways (11), considerable evidence exists to demonstrate that somatosensory evoked potential transmission is essentially dependent upon the integrity of the dorsal columns and reflect activity in neurons sensitive to joint rotation (12-14). Studies in the monkey with segmental dorsal column (DC) resection demonstrated that responses evoked by peripheral nerve or spinal cord electrical stimulation

*Numbers in parentheses designate References at end of paper.

were obliterated at the rostral spinal cord, ventralis posterior lateralis of the thalamus (VPL), and sensorimotor cortex (SMC) (15,16). In contrast, segmental spinal cord ablation with isolated dorsal column preservation resulted in preservation of those responses recorded at the rostral spinal cord, VPL, and SMC levels. However, the response recorded in the nucleus centremedian of the thalamus (CM) was obliterated (16).

For efferent pathway evaluation, stimulation of the motor cortex produces a response which can be measured over the spinal columns reflecting the physiologic integrity of the corticospinal tracts (17). The corticospinal tracts are altered in the same way as the afferent response by cord flexion, distraction or ischemia (8). Consequently, the evoked potentials can be used to evaluate spinal cord dysfunction over afferent or efferent pathways. These studies were, therefore, designed to evaluate changes in the spinal and cerebral afferent and efferent evoked potentials with axial forces applied between the shoulders and skull of the living Macaca mulatta monkey. As part of this study, load deflection curves were obtained from four fresh cadaveric cervical spinal columns of monkeys.

METHODS

EVOKED POTENTIAL - Male monkeys (Macaca mulatta) under thiamylal sodium (10 mg/kg) anesthesia had 0.25 mm diameter bipolar nichrome electrodes, 0.3 mm on center and Formvar covered except at the tip, stereotaxically implanted in VPL or at the junction of VPL and medial lemniscus (VPL-ML). Three in-line, 2 mm diameter platinum discs, 0.0125 mm thick, spaced 4 mm on center, and imbedded in a thin dacron reinforced elastic fiber, were implanted bilaterally over the sensorimotor cortices. Teflon-coated multiple strand 0.0125 mm diameter stainless steel leads from the electrodes were brought out with the other depth electrodes to an Amphenol plug for recording. Methylmethacrylate was used to fill the trepine holes made in the calvarium and for fixation of the recording leads. The three-in-line platinum discs were positioned through small interlaminar openings into the dorsal midline epidural space over the cauda equina (CE) and upper thoracic cord (T2-T4). The thoracic electrodes were positioned for maximum dorsal column response to stimulation of CE electrodes and for maximum efferent volleys secondary to stimulation of electrodes in sensorimotor cortex (SMC). The cord-to-cord responses are defined as the evoked potential recorded at T2-T4 with stimulation of CE, or those responses retrieved at CE with stimulation at T2-T4. The afferent volleys were

maximized at the depth electrodes and SMC electrodes with CE stimulation currents of approximately 0.2 ms duration, 4 Hz from 0.5 to 1.0 mA. The currents were approximately two times that required for the maximum response. For the efferent volleys, currents 6-8 mA, at 0.2 ms duration at 4 Hz were applied to the SMC electrodes on the cortex opposite the SMC recording set. The evoked potentials were recorded with a Clinical Technology Corporation 2000-Evoked Potential Measuring System. On-line recordings of the evoked potential were also made with a Lockheed Store-7 tape recorder. The plots shown for each in vivo study were obtained by measuring the amplitude of the earliest occurring upward or downward evoked potential component. The arrows indicate the beginning of the earliest component. The curves have been smoothed to represent the averaged values over a 2-4 minute interval. Therefore, abrupt changes which occurred with load application are minimized.

FORCE APPLICATIONS AND RECORDINGS - The animals were given a single dose of sodium penthobarbital which lasted throughout the study. A tension force was applied between a yoke fitted over the shoulders and ear bars inserted into the auditory meatus (Fig. 1). In animals 775 through 778, methylmethacrylate was molded into a block incorporating the ear bars and occiput. A modified Baltimore stereotaxic frame which moved smoothly on the rails was used for head fixation and horizontal alignment of the cervical column. A rigid linkage was attached between the ear bars and a Dillon force gauge and a rigid screw firmly fixed to the force gauge. Increments of approximately 25-50 pounds force, 111-222 Newtons (N), were applied every 5-10 minutes. The sled was sufficiently free of friction so that respiratory movements could be recorded on the force gauge. Dial gauge readings were obtained between the ear bars and the yoke. Lateral X-rays at each force level were measured to determine the spacing between centers of each vertebral body. The yoke often prevented initial observation of the lower cervical elements. The tables show the minimum extension of the cervical column. The distraction plotted in the figures and shown in the tables includes the total elongation of the cervical column plus the deflection of the soft tissues beneath the yoke (change in distance from the yoke to the occipital condyles). A 222 N load applied to removed slack was used as the control value. The animals were placed on thermal regulating pads. The core temperature was continuously monitored with a rectal probe. Arterial blood pressures were continuously monitored by a catheter inserted into the femoral artery. Periodic blood gas determinations were made. The animals

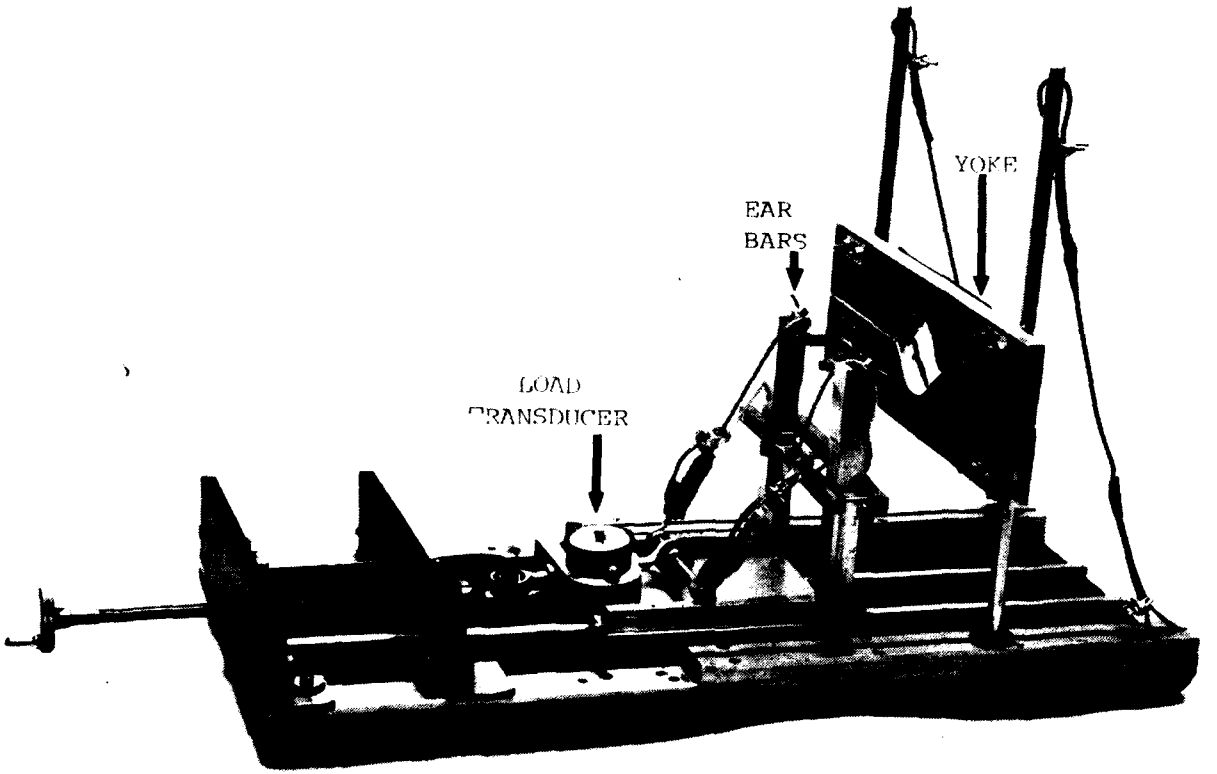

Fig. 1 - In vivo test setup

were intubated and a Harvard Apparatus volume respirator was connected in several animals when the blood gases became abnormal. Heart rate, beats per minute (BPM), was determined from the pressure waveforms. Arteriography was done in selected cases. Gross anatomical inspections were conducted to determine damage. The posterior muscles were sectioned in animals 776 and 777. The animals were sacrificed with an overdose of barbiturate.

ISOLATED CERVICAL COLUMNS - In another study, four naive Macaca mulatta monkeys were sacrificed and the cervical and upper thoracic spinal column, including the base of the skull, was removed. Tissues exclusive of the ligaments were carefully dissected away, and the base of the skull and distal vertebral body were mounted in methylmethacrylate. The specimens were placed in an Instron device. In three monkeys the studies were conducted within 6 hours of sacrifice. In the remaining animal, the studies were conducted 20 hours later. All specimens were kept moist with Ringer's solution prior to and during the experiments. Load deflection curves were obtained for each of the intact specimens and for selected specimens secondary to sectioning of posterior ligaments (all ligaments excluding posterior longitudinal) and anterior ligaments (all ligaments including posterior longitudinal). Photographs were taken at each force level and during large deflections. The Instron vertical loading rate was 0.05 inches per minute. In animals 1 and 2, the first disruption was followed by a second run with the methylmethacrylate applied at the areas shown (Table 1). Failure was considered to occur when the load began to decrease. Failure loads were the maximum values recorded. The acrylic at the base of the skull remained constant in each case. For these animals the skull was mounted in the top fixture which swiveled to allow movement in the horizontal plane, while the fixture carrying the methylmethacrylate of the distal element was fixed to the frame which pulled the spinal column in the vertical direction. For animals 3 and 4, the spinal column was rotated with the movable fixture attached to the distal portion of the specimen and the base of the skull attached to the rigid moving section of the Instron.

RESULTS

ISOLATED CERVICAL COLUMN STUDIES - Disruptions occurred at T1-C7, C6-C7, C4-C5 and C3-C4 with failure loads of 325 N, 534 N, 358 N and 489 N, respectively, for the first runs. The failures always began in the anterior region at the anterior longitudinal ligament

TABLE I

DATA - ALL FRESH RUNS

SPECIMEN	RUN #	NATURE OF PREP.	LOCATION OF FAILURE	% ELONGATION AT FAILURE	FAILURE LOAD N(s)	FAILURE LOAD (LBS)	ENERGY DISSIPATION J	ENERGY DISSIPATION (FT LB)
1 (9.5 kg)	1	(T3)-Base	T1-C7	12.6	325	(73)	2.6	(1.92)
	2	(C7C6)-Base	C4-C5	13.4	347	(78)	1.34	(0.99)
2 (12.5 kg)	1	(T2T1)-Base	C6-C7	24	534	(120)	3.7	(2.75)
	2	(C6C5)-Base	C4-C5	22	400	(90)	1.15	(0.85)
	3	(C4C3)-Base	C2-C1	40	525	(118)	2.0	(1.47)
3 (15.75 kg)	1	(T2T1)-Base	C4-C5	40	358	(80)	2.54	(1.86)
	2	(C4-C3)-Base Ant. Lig. Transected at C3C2 Level	C2-C3	42.5	167	(37.5)	0.59	(0.43)
	3	(C2C1)-Base	C1-Base	86	618	(139)	2.37	(1.75)
	4	(T1C7)-(C6C5) Post. Lig. Transected at C6-C7 Level	C6-C7	38	195	(43.7)	0.46	(0.34)
4 (8.05 kg)	1	(T2T1)-Base	C3-C4	28	489	(110)	2.7	(2.0)
	2	C3-Base	C2-C3	41	423	(95)	1.93	(1.42)
	3	(C2C1)-Base	C1-Base	152	534	(120)	2.5	(1.87)
	4	(T2T1)-(C4C5) Ant. Lig. Transected at C5-C6 Level	C5-C6	32	160	(36)	0.98	(0.72)

NOTE: Runs Monkey 1 & 2 run mounted Base - up.
Runs Monkey 3 & 4 run mounted Base - down.

and disc and preceded posteriorly. Disruption of the flaval ligament, capsular ligament, and interspinous ligament occurred following substantial elongation and at 1/3 to 1/4 the maximum force levels recorded. For the second runs of the complete sections, failures occurred at C4-C5, at 347 N and 400 N. With ablation of the anterior ligaments, failures occurred at C2-C3 at 167 N or C5-C6 at 160 N. Failure was observed with posterior ligament ablation at C6-C7 at 195 N and at C2-C3 with 423 N. Disruptions between C1 and the base of the skull required 534 N and 618 N. The largest percent of elongation prior to failure was observed between C1 and the base of the skull. The energy ranged from 0.46 to 3.7 Joules (J).

Comments - The isolated cervical column studies suggest that the lower cervical region failed first. As the spine was shortened, or sectioned, the failure repeatedly occurred in the lower regions of the remaining specimen. The disruption loads for the C1-base of skull junction were markedly higher, suggesting that the cervical column mechanical strength is a function of the level or location of the vertebrae. Since all disruptions occurred outside the area of fixation, and the jaws of the Instron machine are self-aligning, the effect of fixation was considered negligible.

IN VIVO STUDIES

ANIMAL 777 - The posterior muscles were dissected away from the occiput in this 7.8 kg male. Forces up to 890 N (200 lb force) were applied. Heart rate, elongation of the cervical column, systolic and diastolic blood pressure and evoked potentials recorded at SMC with stimulation of electrodes on CE (CE-SMC) and recordings on the thoracic cord with stimulation of SMC on the opposite side (SMC-T3) are noted (Fig. 2). The cervical column from 0-C5 distended 0.5 cm (Table 2), however, the cervical plus soft tissue movement (distraction) was 1.2 cm (Fig. 2). Blood pressure began to increase at the 667 N load, and the heart rate decreased with application of the 890 N load. Prior to these changes, the amplitude of the early components of the SMC evoked potential began to decrease and was essentially obliterated within 5 minutes following application of the 890 N load (Fig. 3). The efferent response also decreased to zero; however, the changes began approximately 5 minutes after application of the maximum load. Blood gases (pH, pCO_2, pO_2, O_2 saturation) were normal prior to and at 9 minutes following application of the 890 N load.

Analysis of the cervical X-rays (Table 2) show that the largest separations occurred between the occiput and the vertebral body of C1. During the study, the

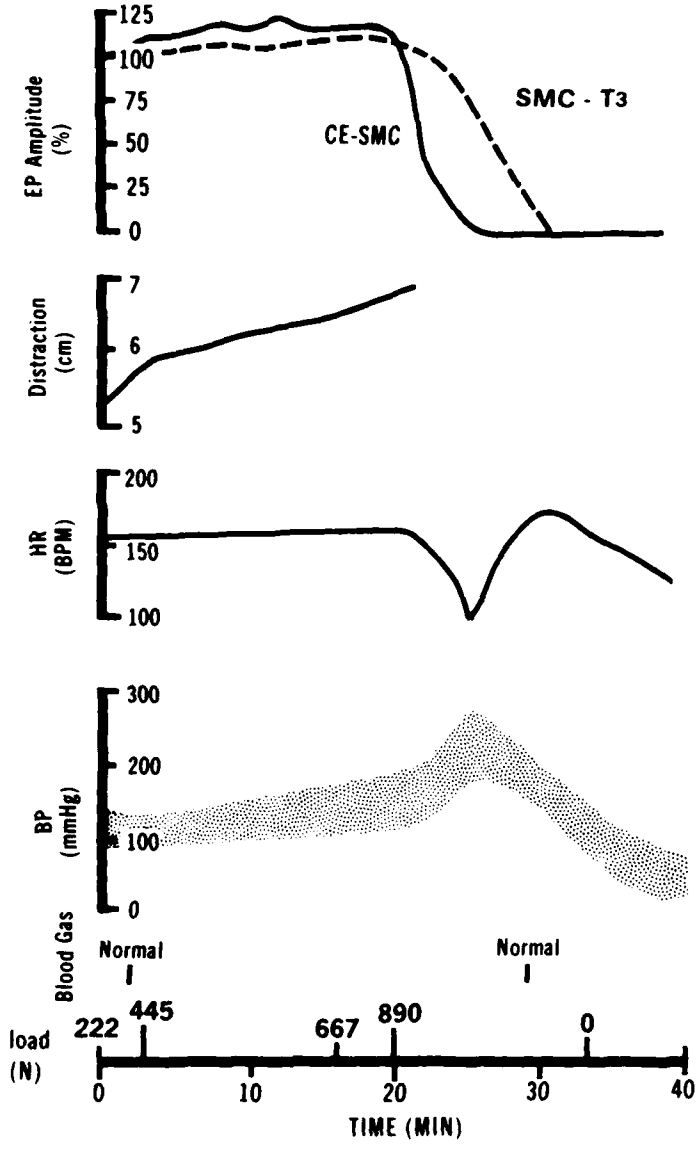

Fig. 2 - Plots of animal #777. (Top) The percentage changes in evoked potential amplitude of the early components of the cauda equina (CE) to sensorimotor cortex (CE-SMC) and sensorimotor cortex to thoracic cord responses (SMC-T3), distraction (soft tissue plus cervical displacement) (Second from top), the heart rate (HR) in beats per minute (Third from top), the systolic and diastolic blood pressure range (Fourth from top) and arterial blood gases versus the applied load in newtons and time in minutes (Bottom)

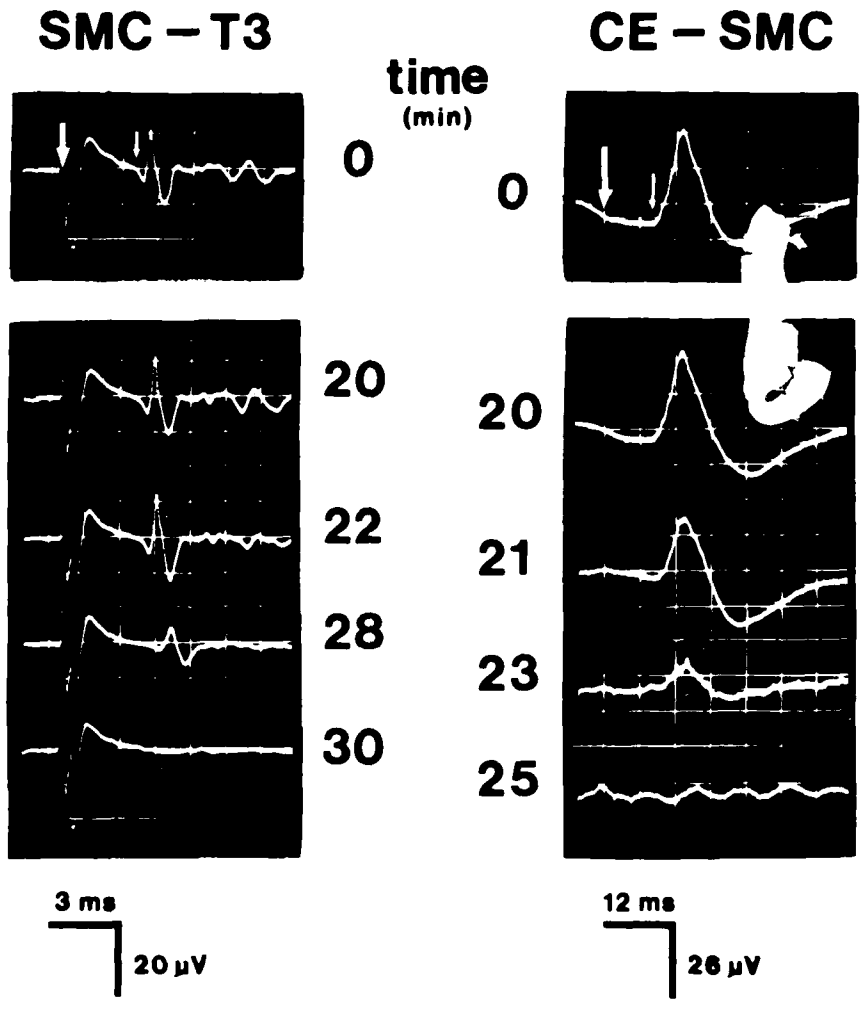

Fig. 3 - Evoked potentials of animal #777. The time in minutes corresponds to the scale of figure 2. Left stimulation of sensorimotor cortex recording at T3 of dorsal column. Right stimulation of cauda equina recording at sensorimotor cortex. Stimulus at first arrow, response onset at second arrow, in all figures

TABLE 2

INTERVERTEBRAL DISTANCE (cm) - Animal #777

LOAD (N)	O-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-YOKE	DISTRACTION+
222	0.5	1.1	1.2	1.4	1.2	0.1	5.3
445	0.6	1.1	1.4	1.3	1.1	0.5	6.0
667	0.7	1.2	1.4	1.4	1.1	0.8	6.5
890	0.8	1.2	1.3	1.4	1.2	0.2*	6.1
Cervical Stretch	0.?	0.1	0.1	0	0	—	

Total Cervical Stretch = 0.5(O-C5)

+Change in distance from occipital condyles to yoke.

*Distance from center of lowest visible vertebral body to yoke.

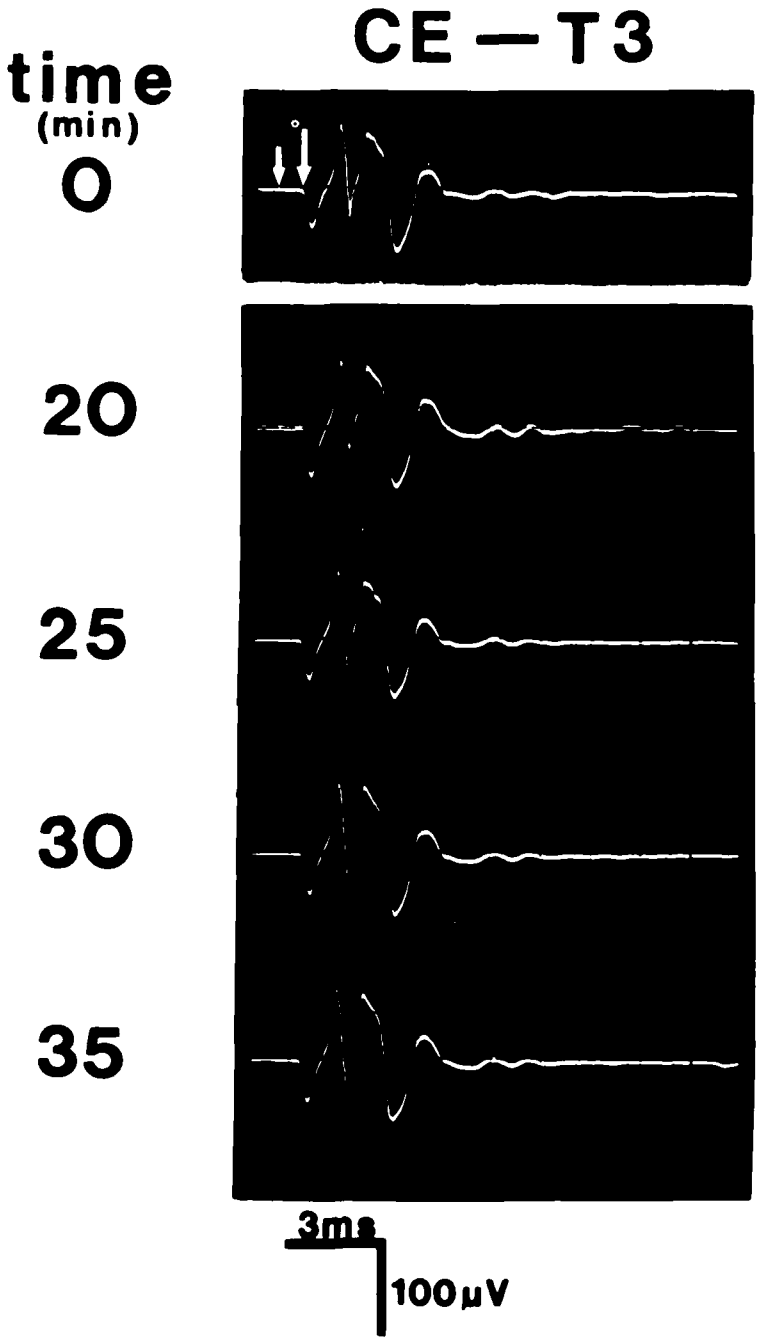

Fig. 4 - Evoked potentials of animal #777. Stimulation at cauda equina with recording at T3 of dorsal column (cord-to-cord)

temperature remained within normal limits. The evoked potentials (cord-to-cord) obtained from the thoracic cord, with stimulation of the CE (CE-T3) were unaltered up until the time of sacrifice (Fig. 4), as were the responses at CE with T3 stimulation. At autopsy, a left vertebral artery disruption and an evulsion of the left articular capsular ligament was observed. The posterior longitudinal ligament was split at C1 through C3. An intradural hematoma was observed at C5-C6. The supraspinous ligament was cut during the muscle sectioning.

Comments - The increase in blood pressure and decrease in heart rate at the higher loads suggest a brainstem phenomenon (Cushing reflex). These changes were concurrent with, or preceded by, reduction in the SMC evoked potential. The later changes in the SMC evoked potential and the efferent response at the thoracic cord were probably due to cerebral ischemia following vertebral artery disruption. In this animal, immediate reduction in the evoked potentials at CE-SMC and SMC-T3 was routinely observed with an increase in force. These changes often returned to control at force levels below 667 N, but were permanent at 890 N. Since the cord-to-cord evoked potentials remained unaltered during these studies, the findings suggest that the changes were probably due to cervical or cerebral effects.

ANIMAL 776 - This 13 kg monkey had the anterior muscles cut with care to preserve the supraspinous and associated posterior ligaments. Good evoked potentials were obtained at VPL, the thalamocrotical pathways and at the SMC with stimulation of CE (CE-SMC). Responses at the thoracic cord (T2) following stimulation of the contralateral cortex were also monitored (SMC-T2). Transient decreases in the SMC evoked potentials occurred with load application. The SMC evoked potentials began to decrease at 1333 N (300 lb force) (Fig. 5). These were followed within several minutes by a reduction in the thoracic cord response (SMC-T2). Responses at VPL and thalamocortical pathways (not shown) began to change 4 minutes later. A slight increase in the VPL response was observed during the early decrease in the SMC response (Fig. 6). All evoked potentials except those at cord-to-cord, began to decrease with the early increases in blood pressure and heart rate. Blood gases were normal 5 minutes following application of the 1333 N load. However, the animal ceased breathing and was placed on the respirator 25 minutes following application of the maximum force. The blood gases demonstrated that the animal was acidotic. Cervical deflections increased to 0.1 cm (0-C7) (Table 3). This animal's blood gases returned to normal with artificial respiration, but was

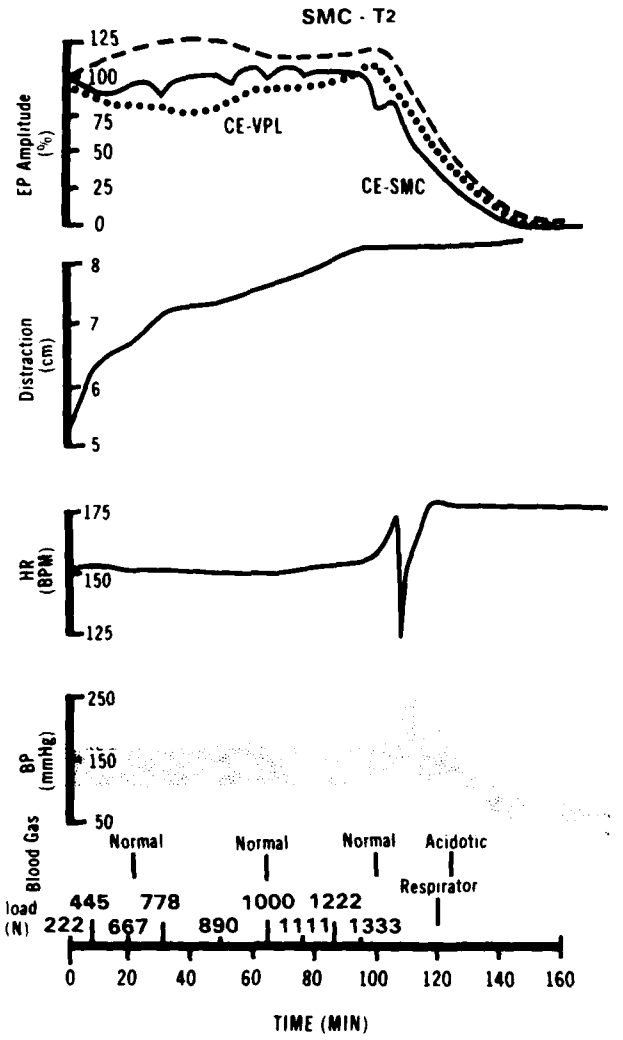

Fig. 5 - Plots of animal #776. (Top) The percentage changes in potential amplitude of early component of sensorimotor cortex to T2 dorsal column recording site (SMC-T2), cauda equina to nucleus ventralis posterior lateralis of the thalamus (CE-VPL), cauda equina to sensorimotor cortex (CE-SMC), distraction (soft tissue plus cervical displacement) (Second from top), the heart rate (HR) in beats per minute (Third from top), the systolic and diastolic blood pressure range (Fourth from top), and arterial blood gases versus the applied load in newtons and time in minutes (Bottom)

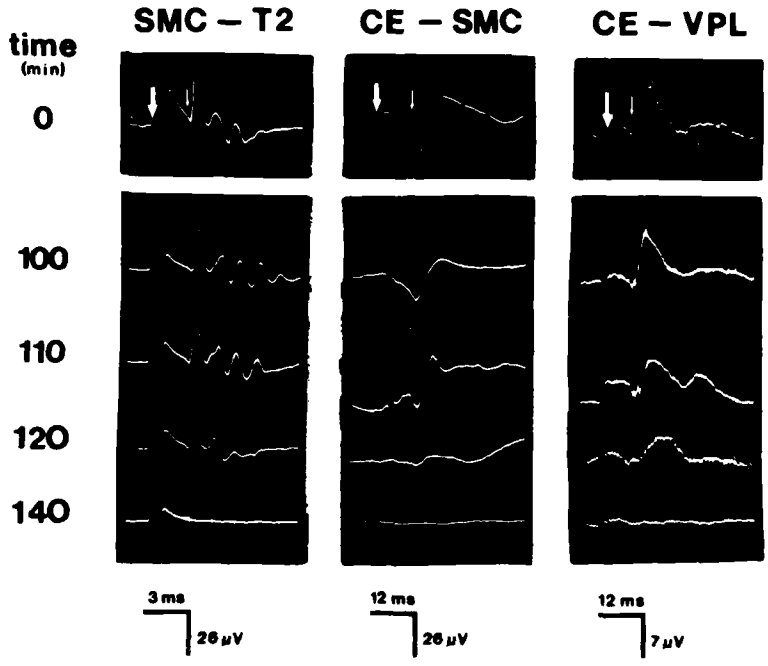

Fig. 6 - Evoked potentials of animal #776

TABLE 3

DEFLECTIONS
Intervertebral Distance (cm) - Animal #776

LOAD (N)	0-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7	DISTRACTION
222	.6	1.1	1.4	1.3	.9*	---	---	5.3
445	.5	1.0	1.3	1.3	1.3	.9*	---	6.3
667	.5	1.0	1.2	1.2	1.5	1.3	0*	6.7
778	.5	1.0	1.3	1.3	1.3	1.3	.3*	7.0
890	.5	1.0	1.3	1.3	1.3	1.4	.6*	7.4
1000	.7	1.0	1.2	1.4	1.3	1.3	.7*	7.6
1111	.6	1.1	1.2	1.5	1.2	1.3	.8*	7.8
1222	.5	1.1	1.3	1.5	1.4	1.2	1.1 .4*	8.5
1333	.5	1.1	1.4	1.5	1.3	1.3	1.2 .7*	8.2
Cervical Stretch	-.1	0	0	.2	0	(0)	(.1)	

Total Cervical Stretch = .1 (0-C7)

Change in distance from occipital condyles to yoke.

*Distance from center of lowest visible vertebral body to yoke.

unable to breath unassisted and was subsequently sacrificed with an overdose of barbiturates. At autopsy a slight capsular disruption at C2-C3 with hemorrhage at the posterior base of the skull in the region of C1, serous fluid and blood over the spinal cord at C1-C2, and subdural blood at C2-C3 was found. The anterior longitudinal ligament appeared very thin and almost translucent, but it was, however, intact along with all other ligaments. While the blood gases returned to normal within approximately 1 hour following application of the 1333 N load, all the evoked potentials, with the exception of those at cord-to-cord, did not. The core temperature of the animal remained within normal limits 40 minutes following application of the maximum load.

Comments - The hemorrhage in the region of C1 to C3 suggests a cervical or brainstem phenomenon and is probably responsible for the changes in blood pressure and heart rate. The immediate changes in the evoked potentials with load application were probably due to mechanical stretching of the cord and brainstem; however, the later changes could be secondary to oxygen deprivation or cerebral circulatory deprivation. The SMC evoked potentials changed prior to the other responses. The depth responses are more resistant to changes as shown by the VPL response. Furthermore, studies in our laboratory have demonstrated that a compensatory mechanism occurs in VPL secondary to stretching of the cord to produce a transitory increase in the amplitude of the VPL evoked potential concurrent with a decrease in the cortical response.

ANIMAL 778 - Up to 1555 N (350 lb force) was applied to an 8.25 kg monkey. Some increases in blood pressure were noted at 1000 N; however, the changes in heart rate were minimal until 1333 N was applied. Evoked potentials at the cortex and thoracic cord began to decrease following application of 1333 N, and were obliterated at 1555 N (Figs. 7,8). The SMC-T2 evoked potentials began to decrease prior to the CE-SMC response. Blood gases were normal and the animal was breathing unassisted. The SMC-T2 and CE-SMC responses returned to approximately 50% of normal amplitude about 30 minutes later. The cervical column increased 0.3 cm (Table 4). A slight trace of blood was observed at the base of the skull in the region of C1. Blood was found along the posterior longitudinal ligament, with a slight disruption of the ligamentum flavum at C1-C2. Traces of blood were found on the cord at C4-C5 and C6-C7. The animal's core temperature remained within normal limits, and the cord-to-cord evoked potentials remained normal up to 20 minutes following obliteration of the CE-SMC and SMC-T2 evoked

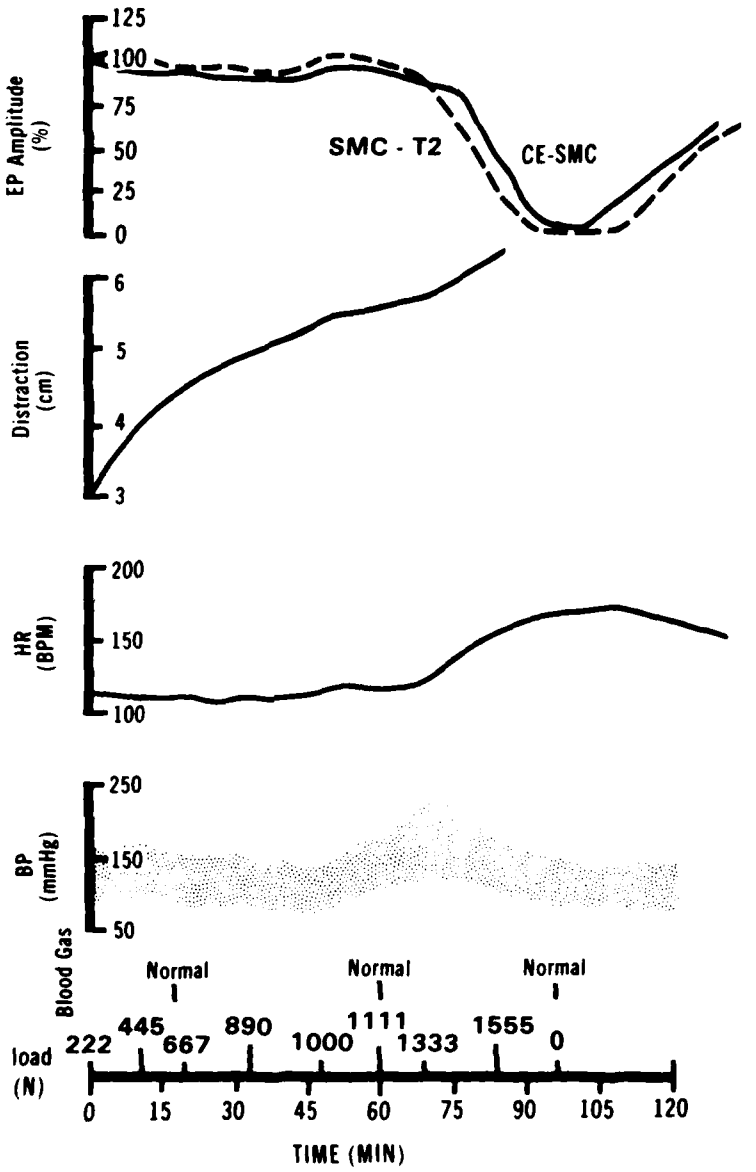

Fig. 7 - Plots of animal #778. (Top) Percentage changes in early evoked potential of CE-SMC and SMC-T2, distraction (soft tissue plus cervical displacement) (Second from top), the heart rate (HR) in beats per minute (Third from top), the systolic and diastolic blood pressure range (Fourth from top) and arterial blood gases versus the applied load in newtons and time in minutes (Bot)

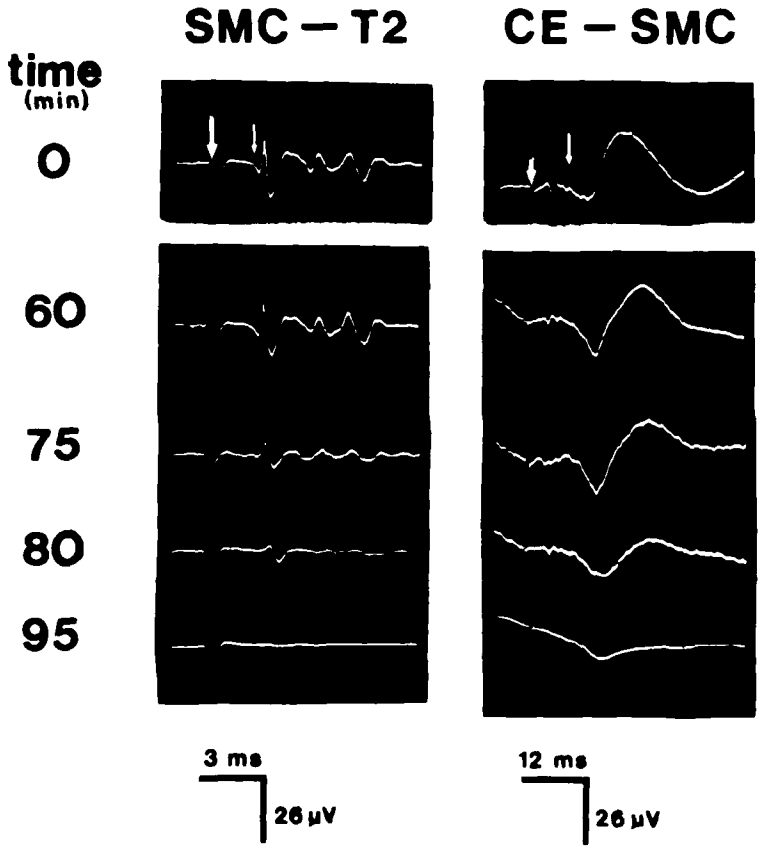

Fig. 8 - Evoked potentials of animal #778

TABLE 4

INTERVERTEBRAL DISTANCE (cm) - Animal #778

LOAD (N)	O-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7	DISTRACTION †
222	.5	1.1	1.0	.2*	---	---	---	2.8
445	.5	1.0	1.0	1.1	.4*	---	---	4.0
667	.5	1.1	1.0	1.0	.9	---	---	4.5
890	.5	1.0	1.1	1.0	1.0	.3*	---	4.9
1000	.6	1.0	1.1	1.1	1.1	.5*	---	5.4
1111	.6	1.0	1.2	1.1	1.1	.6*	---	5.6
1333	.6	1.0	1.2	1.1	1.2	.6*	---	5.7
1555	.6	1.0	1.2	1.1	1.0	1.0	.2*	6.2
Cervical Stretch	+.1	-.1	.2	(0)	(.1)	-		

Total Cervical Stretch = 0.3 (O-C5)

†Change in distance from yoke to occipital condyles.

*Distance from center of lowest visible vertebral body to yoke.

potentials.

Comments - It is difficult to explain the changes in blood pressure without heart rate alteration. However, since the evoked potentials changed while the blood gases remained within normal limits, the alterations were probably due to mechanical factors (Figs. 7,8). The 50% recovery of the evoked potential control levels suggest neural damage.

ANIMAL 775 - A 13 kg monkey had forces up to 1444 N (325 lb force) applied. The evoked potential at the cortex (CE-SMC) began to decrease following 1111 N force application and was approximately 25% of control amplitude at 15 minutes following application of the 1444 N load. The evoked potential changes preceded marked alteration in blood pressure and heart rate (Figs. 9,10, Table 5). Following application of the 1444 N load, the animal required ventilation. Blood gases demonstrated that the animal was acidotic and sodium bicarbonate was given. Within approximately 20 minutes, normal blood gases were obtained, and the evoked potentials recovered to approximately 75% of control. Subsequently, the animal ventilated unassisted. The autopsy showed blood at the base of the skull near the foramen magnum, and blood on the spinal cord at C5-C6. No ligamentous disruption was found. The cord-to-cord evoked potentials and the core temperature were normal.

Comments - The early evoked potential changes were probably due to mechanical factors, however, the later alterations are probably a combination of mechanical and metabolic effects.

ANIMAL 717 - Forces up to 556 N (125 lb force) were applied to a 3.2 kg monkey (Fig. 11). the CE-SMC and CE-VPL evoked potentials began to decrease at 445 N (100 lb force) prior to significant alterations in blood pressure or heart rate (Fig. 12). The cervical column elongated from 1.1 cm (Table 6). A skull fracture at the lambdoidal sutures occurred at a load of 556 N; the animal hemorrhaged through the nose.

Comments - The early changes in the evoked potential preceding the skull fracture were probably due to mechanical factors. The skull sutures were open in this young animal.

ANIMAL 702 - A force up to 1333 N (200 lb force) was applied to an 8.4 kg monkey (Fig. 13 and Table 7). The Leg-SMC evoked potential due to sciatic nerve stimulation at the leg began to decrease with an application of 1222 N during early alterations in blood pressure and heart rate (Figs. 13,14). At 1333 N the animal began to hemorrhage through the nose. The autopsy showed a vertebral artery disruption, tectorial ligamentous damage at the clivus, and posterior longitudinal

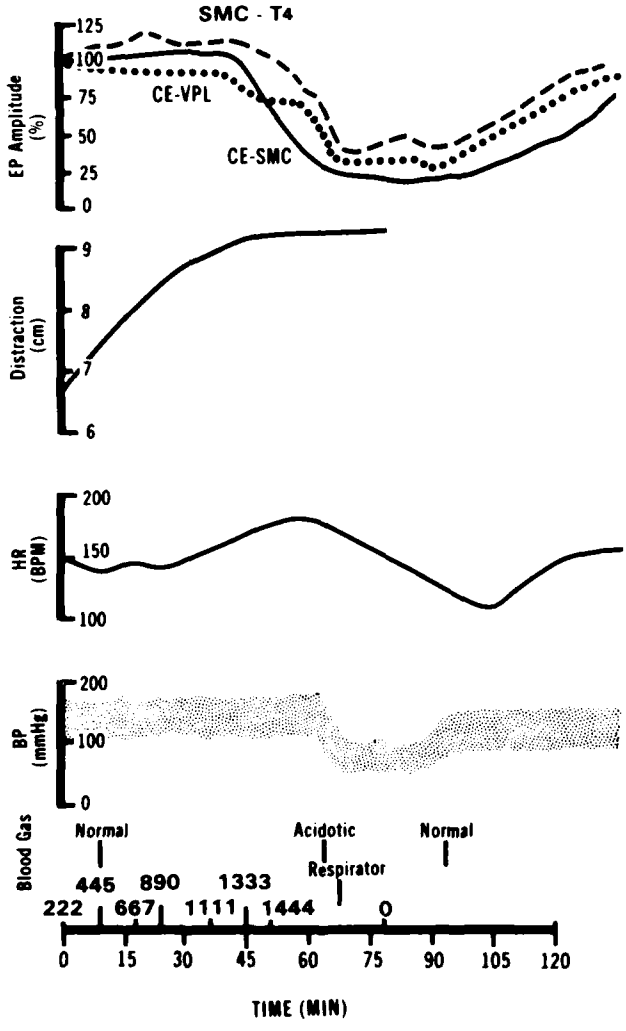

Fig. 9 - Plots of animal #775. (Top) Percentage changes in early evoked potential of SMC-T4, CE-VPL, CE-SMC, distraction (soft tissue plus cervical displacement) (Second from top), the heart rate (HR) in beats per minute (Third from top), the systolic and diastolic blood pressure range (Fourth from top) and arterial blood gases versus the applied load in newtons and time in minutes (Bottom)

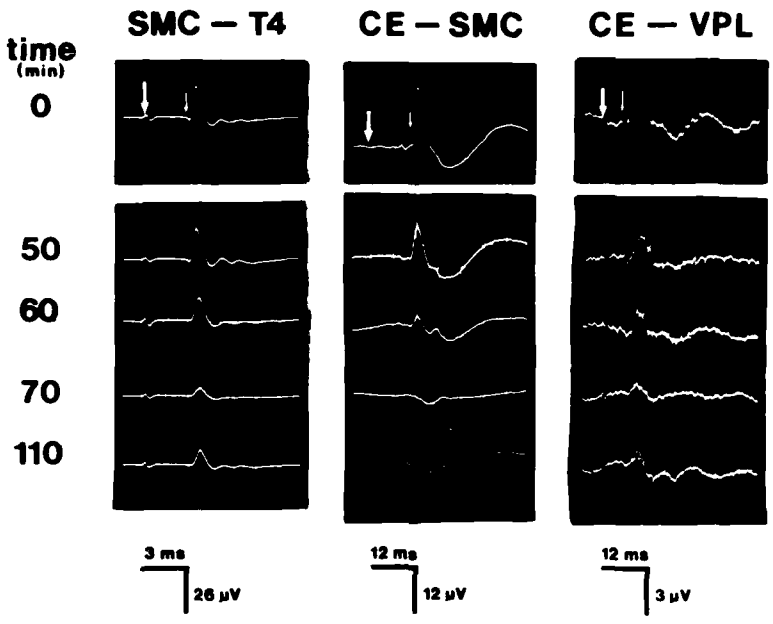

Fig. 10 - Evoked potentials of animal #775

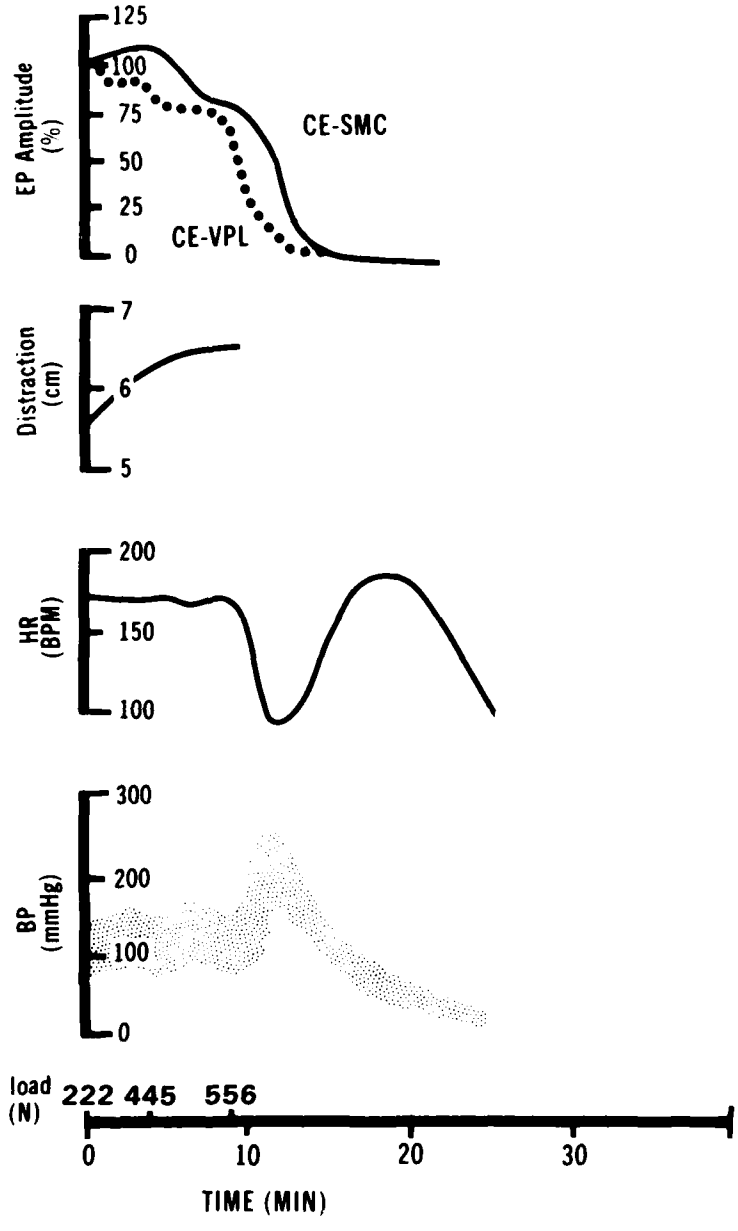

Fig. 11 - Plots of animal #717. (Top) Percentage changes in early evoked potential of CE-SMC, CE-VPL, distraction (soft tissue plus cervical displacement) (Second from top), the heart rate (HR) in beats per minute (Third from top), the systolic and diastolic blood pressure range (Bottom).

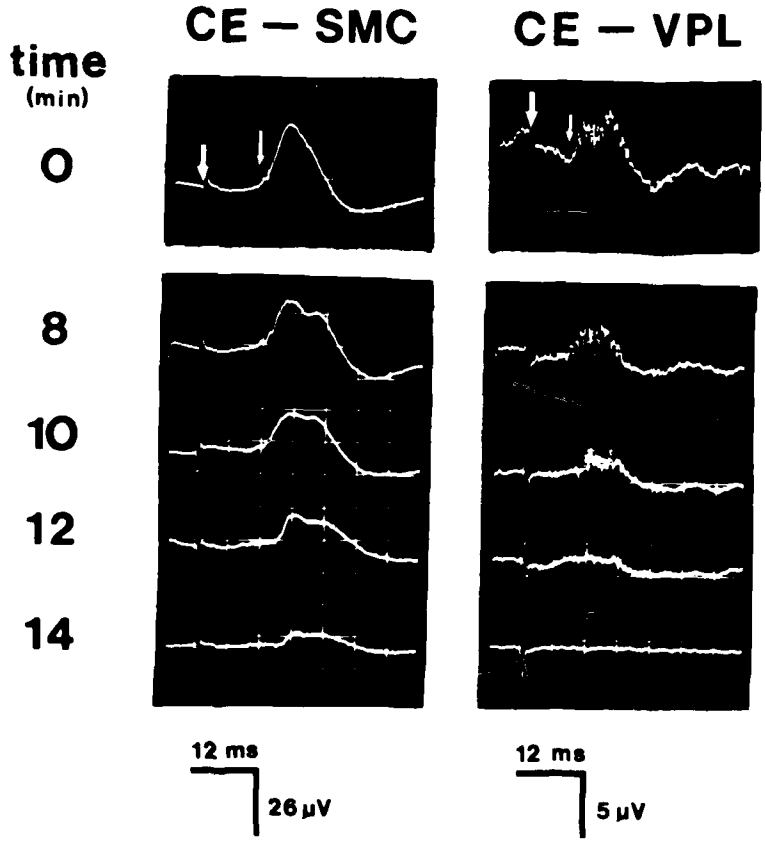

Fig. 12 - Evoked potentials of animal #717

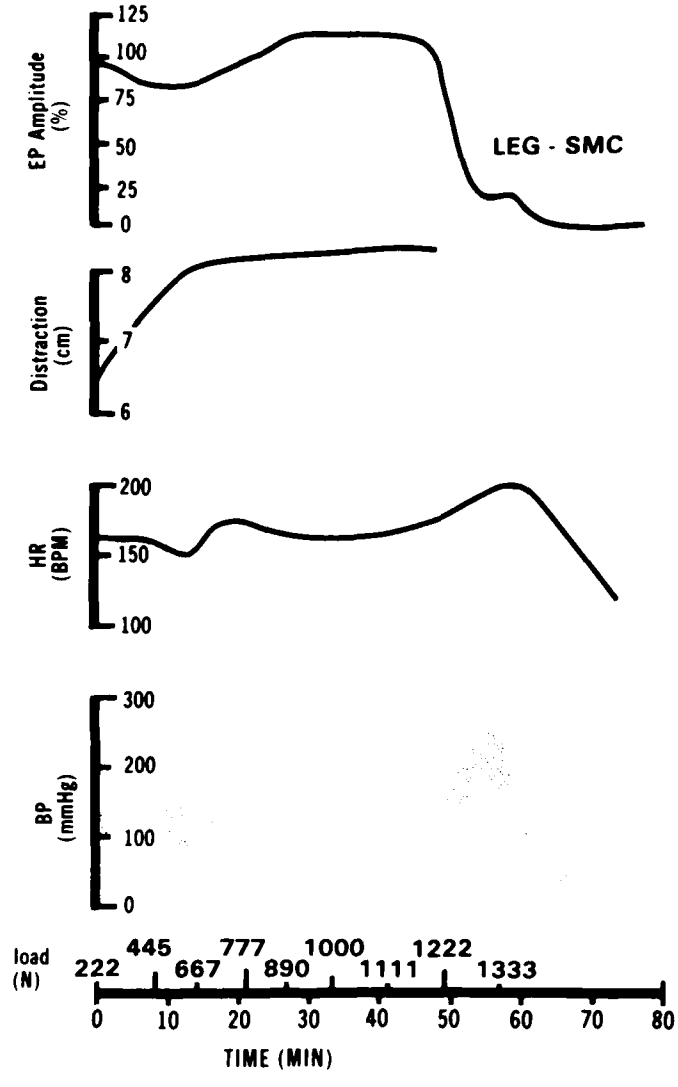

Fig. 13 - Plots of animal #702. (Top) Percentage changes in evoked potential due to stimulation of sciatic nerve at leg with recording at sensorimotor cortex (Leg-SMC), distraction (soft tissue plus cervical displacement) (Second from top), the heart rate (HR) in beats per minute (Third from top), the systolic and diastolic blood pressure range (Bottom).

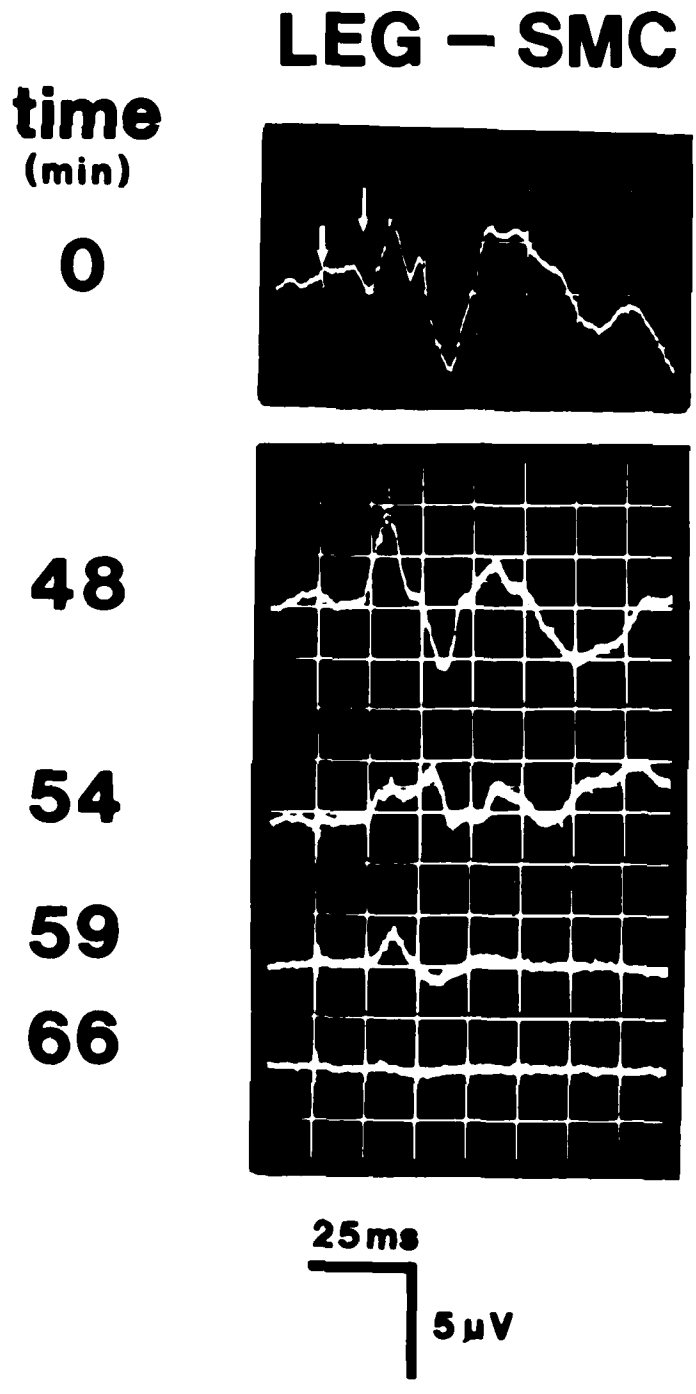

Fig. 14 - Evoked potentials of animal #702

TABLE 5

INTERVERTEBRAL DISTANCE (cm) - Animal #775

LOAD (N)	O-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7	DISTRACTION†
222	.5	1.2	1.6	1.6	1.6	0.1*	---	6.6
445	.5	1.1	1.6	1.6	1.5	1.2*	---	7.5
667	.5	1.3	1.5	1.6	1.4	1.7*	0.2*	8.0
890	.5	1.4	1.6	1.5	1.3	1.5	0.6*	8.4
1111	.6	1.4	1.6	1.6	1.3	1.4	1.0*	8.9
1333	.5	1.4	1.7	1.6	1.4	1.4	1.2*	9.2
1444	.6	1.4	1.6	1.6	1.4	1.3	1.3*	9.2
Cervical Stretch	.1	.2	0	0	(-.1)	(-.2)	-	

Total Cervical Stretch = 0 (O-C6)

†Change in distance from yoke to occipital condyles.

*Distance from center of lowest visible vertebral body to yoke.

TABLE 6

INTERVERTEBRAL DISTANCE (cm) - Animal #717

LOAD (N)	O-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7	C7-YOKE	DISTRACTION†
222	.7	.8	.8	.8	.8	.8	.8	-	5.5
445	.7	.9	1.2	.9	.9	.9	.9	.3*	6.7
556	.7	1.1	1.2	.9	.9	.9	.9	.5*	7.0
Cervical Stretch	0	.3	.4	.1	.1	.1	.1	0	

Total Cervical Stretch = 1.1 (O-C7)

†Change in distance from occipital condyle to yoke.

*Distance from center of lowest visible vertebral body to yoke.

TABLE 7

INTERVERTEBRAL DISTANCE (cm) - Animal #702

LOAD (N)	O-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7	C7-YOKE	DISTRACTION†
222	.5	0.9	1.3	1.3	1.1	1.2*	---	---	6.3
445	.5	1.0	1.4	1.3	1.1	1.2	1.05*	---	7.55
667	.6	0.9	1.5	1.3	1.15	1.2	1.5*	---	8.15
777	.6	1.0	1.5	1.3	1.2	1.2	1.5	0.5*	8.7
890	.5	1.0	1.4	1.3	1.2	1.3	1.5	0.7*	8.9
1000	.5	1.2	1.4	1.3	1.2	1.3	1.4	1.0*	9.3
1111	.5	1.2	1.4	1.4	1.2	1.3	1.4	1.8*	10.2
1222	.5	1.2	1.3	1.4	1.2	1.3	1.5	1.7*	10.1
Cervical Stretch	0	.3	0	.1	.1	.1	0		

Total Cervical Stretch = 0.6(O-C7)

†Change in distance from occipital condyle to yoke.

*Distance from center of lowest visible vertebral body to yoke.

ligament disruption at C5-C7. Core temperature was normal.

Comments - The decrease in Leg-SMC evoked potentials which was preceded by the vascular failure, was probably caused by mechanical stretching of the cervical cord or brainstem.

ANIMAL 722 - This 4 kg male monkey had forces applied up to 867 N (195 lb force). At 667-778 N (150 - 175 lb force), a reduction in heart rate from 175 to 72 BPM was observed with no change in the SMC evoked potential with sciatic nerve stimulation. At 867 N a loud snap was heard, the load dropped to 422 N, the heart rate returned to control levels, but the animal required ventilation. The evoked potential was progressively obliterated over a 15 minute period. Angiograms indicated no gross cerebral vessel damage. At autopsy C1-C2 had serous exudate with posterior ligamentous disruption. All other ligaments were intact.

Comments - The changes in the evoked potential suggest spinal or circulatory dysfunction was probably involved since the response deteriorated over approximately 15 minutes. Cord-to-cord evoked potentials or blood gases were not available.

ANIMAL 732 - This 5.5 kg monkey sustained a fracture at the lambdoidal sutures with a force application of 978 N (220 lb force). At 667 N the heart rate decreased from 130 to 80 BPM, but increased to 200 BPM at 778 N. At 978 N a large deflection was noted, the load dropped to 623 N, and the animal hemorrhaged through the nose. The SMC evoked potentials due to sciatic nerve stimulation were obliterated within 5 minutes.

Comments - Cerebral ischemia due to the skull fracture is the probable cause of evoked potential reduction. Blood gases or cord-to-cord evoked potentials were not available.

DISCUSSION

The evoked potentials recorded from somatosensory cortex and at the dorsal column of the spinal cord due to peripheral stimulation are reversibly reduced in amplitude both by pathological distraction and flexion of the vertebral column (8). Furthermore, afferent cerebral evoked responses and efferent spinal cord responses to cortical stimulation are obliterated within two minutes following occlusion of the ascending aorta, while the cord-to-cord responses persist without change for approximately ten minutes and then gradually disappear. Since the cord-to-cord responses were unaltered in these studies, the cerebral circulation was probably

intact. However, cerebral metabolic studies are required to substanciate this hypothesis. In stretch studies conducted on a single frog axon, conduction obliteration has been attributed to changes in resistance of the axis cylinder (18). Since the afferent and efferent evoked potentials usually changed immediately with load application, the reduction in the early components may be due to axonal stretch. While stretching of the carotid and vertebral arteries could be a factor, the minimal cervical distractions probably preclude this effect. Furthermore, the early components of the afferent SMC or depth evoked potentials recorded with either peripheral nerve or dorsal column stimulation at cauda equina, generally began to reduce prior to marked alteration in heart rate or blood pressure at the higher force levels used in this study. The efferent evoked potentials recorded at thoracic spinal cord due to sensorimotor cortex stimulation usually followed the afferent evoked potentials, but were, however, also sensitive indicators of changes in vital functions. The general rule was that the evoked potential changes preceded or changed with major alterations in vital functions. While vertebral artery disruption was observed in two animals, only minor ligament damage was observed in all the in vivo studies. The removal of the posterior muscles in the 2 animals did not demonstrate conclusive differences in failure levels, particularly since animal 777 was found to have vertebral artery disruption at 890 N (200 lb force) which may have been secondary to destruction of some upper posterior ligaments. In contrast, the other animal (776) whose posterior muscles were transected reached terminal force application of 1333 N (300 lb force). The younger animals withstood smaller force applications than the larger ones. Studies in a human spinal column of a 79.5 kg unembalmed 50-yr old male cadaver showed a C4-C5 disruption at 1446 N of axial force. The separation commenced in the anterior region and proceeded posteriorly as in the monkey preparations. In another human spinal column of a 53 kg unembalmed 36-yr old male cadaver, disruption of the posterior ligaments of C4-C5 occurred at 622 N with ablation of the anterior ligaments, and at 1289 N with the posterior ligaments of C2-C3 transected.

Figure 15 shows the load deflection curves for a typical isolated spinal column study and an in vivo experiment. The isolated spinal column studies demonstrate that the tissues adjacent to the intact spinal column probably carry approximately two times the load of the spinal column. While it was not possible to determine the extrusion of the brainstem through the foramen magnum, it has been suggested as the mechanism for failure (19-23). These preliminary ex-

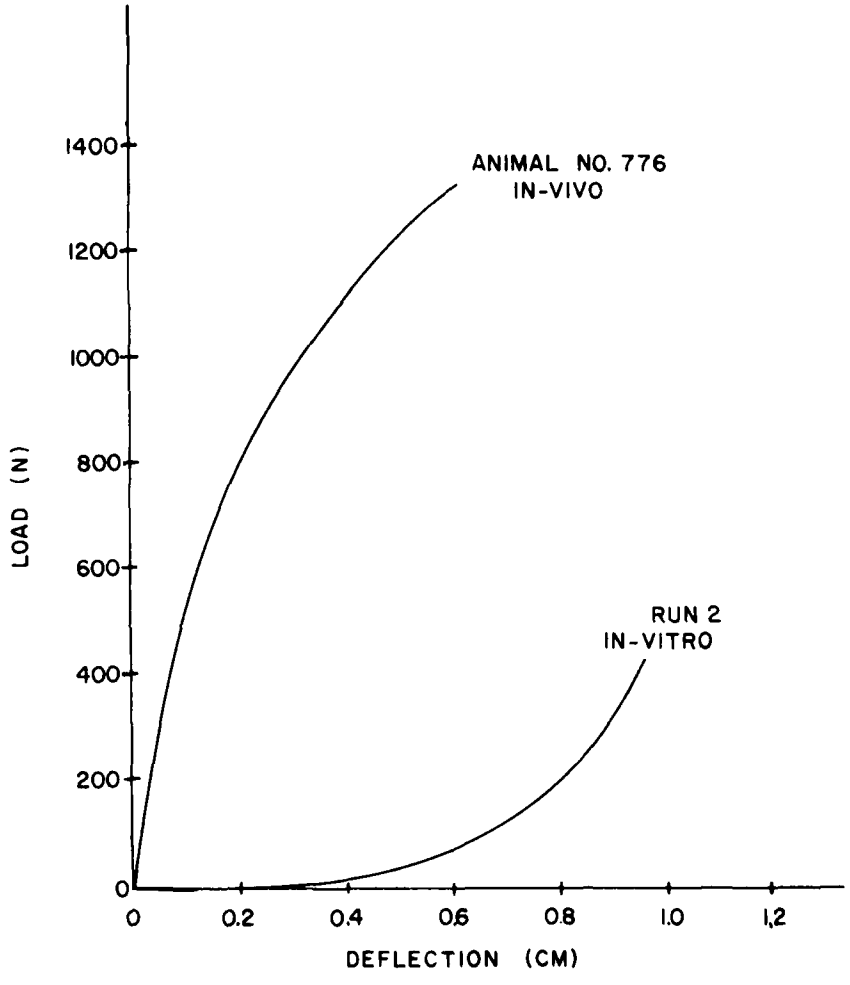

Fig. 15 - Load deflection curves for in vivo and in vitro experiments from base of skull to C-5, specimen 2, run 2 (Table 1)

perimental findings suggest that the evoked potential is an indicator of physiologic changes secondary to application of axial forces at the cervical level, and may be useful for the evaluation of neurologic alterations at the spinal and cerebral levels for subjects exposed to impact or inertial accelerations (17,24,25).

ACKNOWLEDGEMENT

This research was supported in part by the Office of Naval Research Contract N00014-77-C-0749.

REFERENCES

1. A. Sances, Jr., J. Myklebust, S.J. Larson and J.F. Cusick, "The evoked potential and early studies of bioelectricity." J Clin Eng 5(1):27-32, January-March, 1980.

2. J.J. Ackmann, S.J. Larson, A. Sances, Jr. and R.E. Barr, "Non-invasive monitoring techniques in neurosurgical intensive care." J Clin Eng 4(4):329-337, 1979.

3. R.P. Greenberg, D.J. Mayer, D.P. Becker and J.D. Miller, "Evaluation of brain function in severe human head trauma with multimodality evoked potentials. Part I: Evoked brain-injury potentials, method, and analysis." J Neurosurg 47:150-162, 1977.

4. R.P. Greenberg, D.P. Becker, J.D. Miller and D.J. Mayer, "Evaluation of brain function in severe human head trauma with multimodality evoked potentials. Part II: Localization of brain dysfunction and correlation with posttraumatic neurological condition." J Neurosurg 47:163-177, 1977.

5. S.J. Larson, A. Sances, Jr., J.J. Ackmann and D.H. Reigel, "Non-invasive evaluation of head trauma patients." Surgery 74:34-40, 1973.

6. A. Sances, Jr., S.J. Larson, J.F. Cusick, J. Myklebust, C.L. Ewing, R.W. Jodat, J.J. Ackmann and P.R. Walsh, "Early somatosensory evoked potentials." Electroencephalogr Clin Neurophysiol 45(4):505-514, 1978.

7. A. Sances, Jr., J. Myklebust, S.J. Larson, J.F. Cusick and P.R. Walsh, "Theoretical Investigations and Clinical Application of the Evoked Potential." IEEE Frontiers of Engineering in Health Care, Denver, Colorado, pp. 175-179, Oct. 6-7, 1979.

8. S.J. Larson, P.R. Walsh, A. Sances, Jr., J.F. Cusick, D.C. Hemmy and H. Mahler, "Evoked potentials in experimental myelopathy." Spine (In Press).

9. S.J. Larson, R.A. Holst, D.C. Hemmy and A. Sances, Jr., "The lateral extracavitary approach to traumatic lesions of the thoracic and lumbar spine." J Neurosurg 45:628-637, 1976.

10. J.M. Singer, G.V. Russell and J.E. Coe, "Changes

in evoked potentials after experimental cervical spinal cord injury in the monkey." Exp Neurol 29:449-461, 1970.

11. W.T. Liberson, M. Gratzer, A. Zales and B. Wrabinski, "Comparison of conduction velocities of motor and sensory fibers determined by different methods." Arch Phys Med 47:17-23, 1966.

12. D.R. Giblin, "Somatosensory evoked potentials in healthy subjects and in patients with lesions of the nervous system." Ann NY Acad Sci 112:93-142, 1964.

13. A.M. Halliday and G.S. Wakefield, "Cerebral evoked potentials in patients with disassociated sensory loss." J Neurol Neurosurg Psychiat 26:211-219, 1963.

14. S.J. Larson, A. Sances, Jr. and P.C. Christenson, "Evoked somatosensory potentials in man." Arch Neurol (Chic.) 15:88-94, 1966.

15. J.F. Cusick, J. Myklebust, S.J. Larson and A. Sances, Jr., "Spinal evoked potentials in the primate: Neural substrate." J Neurosurg 49:551-557, 1978.

16. J.F. Cusick, J. Myklebust, S.J. Larson and A. Sances, Jr., "Spinal cord evaluation by cortical evoked responses." Arch Neurol 36(3):140-143, 1979.

17. P.R. Walsh, S.J. Larson, A. Sances, Jr., C.L. Ewing, D.J. Thomas, M. Weiss, M. Berger, J. Myklebust, J.F. Cusick and B. Saltzberg, "Experimental methods for evaluating spinal cord injury during impact acceleration." In Electrotherapeutic Sleep and Electroanesthesia, F.M. Wageneder, et al, eds., Universitat Graz, 1978, pp. 435-443.

18. J.A.B. Gray and J.M. Ritchie, "Effects of stretch on single myelinated nerve fibers." J Physiol 124:84-99, 1954.

19. R. Friede, "Specific cord damage at the atlas level as a pathogenic mechanism in cerebral concussion." J Neuropathol Exp Neurol 19:266-279, 1960.

20. R. Friede, "Experimental concussion acceleration." Arch Neurol 4:449-462, 1961.

21. R. Friede, "The Pathology and Mechanics of Experimental Cerebral Concussion." Wadd Technical Report 61-256, Air Research and Development Command, United States Air Force, Wright-Patterson Air Force Base, Ohio, 1961.

22. C.L. Ewing and F. Unterharnscheidt, "Neuropathology and cause of death in U.S. naval aircraft accidents." AGARD Conf Proc No. 190 *on* Recent Experience/ Advances in Aviation Pathology, 7 Rue Ancelle 92200 Neuilly Sur Seine, France, pp. B16-1 to B16-6.

23. C.L. Ewing, "Injury criteria and human tolerance for the neck." In Aircraft Crashworthiness, K. Saczalski et al, eds., University Press of Virginia, Charlottesville, 1975, 11 pp.

24. M.D. Berger, M.S. Weiss, A. Sances, Jr., P.R. Walsh and S.J. Larson, "Evaluation of changes in CNS function due to impact acceleration." Proc Aerospace Med Assoc 50th Ann Scientific Mtg, Washington, D.C., May 14-17, 1979, pp. 135-136.

25. A. Sances, Jr., J.B. Myklebust, S.J. Larson, J.F. Cusick, R.C. Weber and P.R. Walsh, "Bioengineering Analysis of Head and Spine Injuries." CRC Crit Rev Bioeng (In Press).